Medical Student Poster Competition

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Premature Aortic And Carotid Stiffness in Systemic Lupus Erythematosus: Which One Is First or Worse?

Background: Increased aortic and carotid stiffness is common in patients with systemic lupus erythematosus (SLE) and is associated with increased morbidity and mortality. Although the aorta and carotid arteries have different anatomy, hemodynamics, and cardiovascular disease implications, it is unknown whether stiffness affects these arterial beds in a parallel fashion and similar extent or with a regional difference.

Methods: 43 SLE patients (41 women, age 36±11 years) and 17 age-and-sex matched healthy volunteers (14 women, age 34±12 years) underwent clinical and laboratory evaluations and transesophageal echocardiography and carotid ultrasonography for assessment of aortic and carotid stiffness, respectively. Stiffness at the proximal, mid, and distal thoracic aorta and stiffness of the right common carotid artery were assessed using a standard Pressure-Strain Elastic Modulus (PSEM) s=k(sBP−dBP)/[(sD−dD)/dD] where k = conversion factor into Pascal units, sBP=systolic blood pressure, dBP=diastolic blood pressure, sD=systolic diameter, and dD=diastolic diameter. Results. Aortic stiffness was higher in SLE patients than in controls (7.96±4.16 versus 5.76±2.32 units, p=0.01). However, carotid stiffness was not significantly higher in patients versus controls (6.39±2.70 versus 5.55±2.68 units, p=0.29). In SLE patients, aortic stiffness was higher than carotid stiffness (7.96±4.17 versus 6.39±2.70 units, p=0.02). Aortic stiffness also occurred earlier and greater than carotid stiffness in relation to age at diagnosis of SLE (p=0.03).

Conclusions: 1) In young adult SLE patients, aortic stiffness is higher than in matched healthy controls; and 2) in SLE patients, aortic stiffness is higher and may precede carotid stiffness. These findings suggest an accelerated and different pathogenesis of aortic as compared to carotid stiffness in SLE.
**Hyperhemolysis without Hemoglobinopathy- An Unusual Presentation**

**Introduction:** Hyperhemolysis is characterized by a hemolytic transfusion reaction leading to a life-threatening anemia with a drop in hemoglobin and hematocrit (H&H) to levels markedly lower than were present before transfusion. This is a rare occurrence in patients without hemoglobinopathies. Case reports are usually associated with sickle cell anemia or thalessemia. This case highlights hyperhemolysis secondary to a delayed blood transfusion reaction.

**Case Report:** 55 year old male with PMH of hypertension and hyperlipidemia sustained multiple fractures after a complex motorcycle accident. He received 10 units of blood for active bleeding during orthopedic surgery. H/H at time of discharge was 8.2/24. Seven days later after discharge, he returned to the ED with progressive dyspnea, nausea and non-bloody emesis. H&H on presentation was 4.6 and 12 a Hgb drop of 56%, WBC at 64.2K, LDH at 2355, total bilirubin at 5.9, indirect at 4.3, and haptoglobin <8, urinalysis positive for hemoglobin. Delayed hemolytic transfusion reaction was confirmed with positive DAT (IgG and complement) and, with Jka antibody isolated as the cause. He became severely symptomatic with H&H at 5.4 and 15, and was transfused with 2 units of Jka negative blood. His H&H initially rose to 6.1 and 16, but sharply dropped to 5.0 and 14. The next day, the H&H had dropped further to 4.6 and 13, and he was transfused again with 1 unit. Again, his H&H rose directly after transfusion to 5.8 and 17, but dropped to 5.4 and 15. He received another unit with subsequent H&H values of 5.3 and 15. Further transfusions were held. Active bleeding was ruled out with negative guaiac, non-significant nasogastric tube findings, and normal abdominal CT. Further workup ruled out glucose-6-phosphate dehydrogenase or pyruvate kinase deficiency, or any underlying hemoglobinopathy. H&H was 6.1 and 19, with a normal WBC at time of discharge.

**Discussion:** Limited cases reports demonstrate hyperhemolysis with myelofibrosis and anemia of chronic disease, suggesting that hyperhemolysis can occur in the absence of underlying hemoglobinopathies. Our patient developed a delayed hemolytic transfusion reaction, and his hemoglobin and hematocrit dropped to levels lower than pretransfusion with subsequent transfusions, consistent with hyperhemolysis. This case further demonstrates the possibility of hyperhemolysis in patients without hematologic disease; diagnosis of this syndrome should not be dismissed in such patients without a hemoglobinopathy.
Calcific Valvular Disease in End-Stage Renal Disease Patients

It is well known that end stage renal disease (ESRD) patients, especially those on hemodialysis, are prone to coronary complications, at least partially due to advanced calcification of coronary vessels. These patients are more likely to have coronary artery disease (CAD), silent myocardial ischemia, complex ventricular arrhythmias, atrial fibrillation, left ventricular hypertrophy and other complications. There is less awareness among healthcare practitioners of valvular calcification leading to significant morbidities.

We present the case of an ESRD patient on hemodialysis (HD) who was admitted to our hospital with exertional shortness of breath when climbing the stairs of his apartment home. His shortness of breath was initially thought to be due to pneumonia. Eventually the cause of his dyspnea was found to be congestive heart failure (CHF), pulmonary hypertension and atrial fibrillation secondary to a calcified stenotic mitral valve. He underwent cardiac catheterization and was found to have calcification in all vessels, as well as moderate pulmonary hypertension, and severe mitral stenosis. The patient was treated for CHF and discharged with improvement in his symptoms. He continues to follow up at cardiology “valvular disease” clinic.

Valvular calcification itself is very common in end stage renal disease, occurring in up to 30% of patients. However only about 13% of ESRD patients with cardiac calcifications develop atrial fibrillation and in one study up to 40% had thickening of the mitral valve leading to hemodynamic abnormalities. Despite the frequency of calcifications, this important indicator is often overlooked. Extent of calcification can be used as a measure of risk for inducible myocardial ischemia when observed near the induction of dialysis. Currently regular screening for cardiac calcifications is not frequently performed in ESRD. The high rates of hemodynamically significant calcific mitral disease with possible progression to CHF in ESRD/HD patients is often under acknowledged. Appropriate screening of ESRD/HD patients is important for early diagnosis and appropriate follow up and treatment prior to progression of this condition. Unusual complications, such as in this case may be observed if early intervention does not occur.
Growing Old, Not Up: The Juxtaposition of Adult and Pediatric Medicine in Cornelia de Lange Syndrome

Introduction: Cornelia de Lange Syndrome (CdLS) is a genetic disorder affecting approximately 1:10,000 children. Mutations are most commonly found on the NIPBL gene on chromosome 5p13. Cardiac, GI, ophthalmic, musculoskeletal, and immune systems abnormalities may be seen. Death most frequently occurs from aspiration pneumonia in the first 2-4 years of life; however, survival beyond the 5th decade is reported.

Case Presentation: A 40 year old, 16 kg male presented with four days of increasing lethargy and oral intolerance. He was diagnosed with CdLS at birth. An identical twin brother, also with CdLS, died at age 18 from aspiration pneumonia. A history of esophageal strictures led to EGD dilations and undescended testicles resulted in bilateral orchiectomy. All teeth were removed at age 35 for persistent discomfort. Baseline diet included juice, soup, and pediasure. His normal behavior involved nonverbal interaction with his elderly parents, crawling and playing with toys. His exam showed microcephaly, gross limb deformities in hands and feet, and scoliosis. His sodium was 158. He was admitted for rehydration and EGD to the adult hospital medicine service after being refused by the pediatric service due to his age. Correcting his free water deficit required consultation from pediatrics with repeated fluid rate adjustments given his small size. Urine output could not be measured as diaper scales were not available for adult medicine. IV and PICC line placement was also difficult, as his dimensions and behavior were child like, and the placement teams were not trained for children. A crib had to be borrowed from the pediatric service. The crash cart had to be restocked with pediatric sized blades, tubes, and other equipment. His sodium and volume status were eventually corrected. EGD revealed esophageal stricture and candidiasis requiring two dilatations and fluconazole. After 14 days he was discharged.

Discussion: This case begs for discussion of the definitions of pediatric and adult medicine. This 16 kg, 40 year old male was admitted to an adult medicine service that was not prepared to meet the needs of a child-sized adult. His age excluded him from pediatric floor, nursing care, and anesthesia. His parents initially felt like he was rejected by both adult and pediatric services. His case is one example where arbitrary designation of “adult” based on age resulted in prolonged hospital stay, and at least initially, lower quality of care. Management of this patient required coordination between adult and pediatric services which seems counterintuitive.
From Home Deliveries to Health Care Facilities: Establishing a Traditional Birth Attendant Referral Program in Kenya

Objective: To assess the effectiveness of a traditional birth attendant (TBA) referral program on increasing the number of deliveries overseen by skilled birth attendants (SBA) in rural Kenyan health facilities before and after the implementation of a free maternity care policy.

Methods: In a rural region of Kenya, TBAs were recruited to educate pregnant women about the importance of delivering in healthcare facilities and were offered a stipend for every pregnant woman whom they brought to the healthcare facility. We evaluated the percentage of prenatal care (PNC) patients who delivered at the intervention site compared with the percentage of PNC patients who delivered at rural control facilities, before and after the referral program was implemented, and before and after the Kenya government implemented a policy of free maternity care. The window period of the study was from July of 2011 through September 2013, with a TBA referral intervention conducted from March to September 2013.

Results: The absolute increases from the pre-intervention period to the TBA referral intervention period in SBA deliveries were 5.7% and 24.0% in the control and intervention groups, respectively (p < 0.001). The absolute increases in SBA delivery rates before the implementation of the free maternity care policy were 4.7% and 17.2% in the control and intervention groups, respectively (p <0.001). After the policy implementation the absolute increases were 1.8% and 11.6% in the control and intervention groups, respectively (p <0.001).

Conclusion: The percentage of SBA deliveries at the intervention health facility significantly increased compared to control health facilities when TBAs educated women about the need to deliver with a SBA and when TBAs received a stipend for bringing women to local health facilities to deliver. Furthermore, this TBA referral program proved to be far more effective in the target region of Kenya than a policy change to provide free obstetric care.
A Case of a Very Complex Seizure

Introduction: Posterior reversible encephalopathy syndrome (PRES) is a rare condition characterized by seizures, visual disturbances, altered mental status and headache in association with neuroimaging findings of cerebral edema. The exact incidence of PRES is unknown, but it has been increasingly reported in the literature. The pathogenesis of PRES is complex, and incompletely understood. It has been associated with several distinct etiologies, including hypertensive encephalopathy, eclampsia, use of immunosuppressive drugs, and autoimmune disease. Only a small number of cases have been documented among patients with systemic sclerosis. We present the case of a patient with systemic sclerosis who presented with altered mental status, seizures, and hypertensive urgency, and ultimately diagnosed with PRES.

Case description: A 62 year old woman with a past medical history of systemic sclerosis, hypertension, Raynaud phenomenon, GERD, and small bowel pseudo-obstructions with recent small bowel resection, presented from an outside facility following tonic-clonic seizures and altered mental status. Vital signs were notable for tachycardia and hypertension with elevated pulse pressure. She was alert, but confused. Physical exam was remarkable for bilateral sclerodactyly, telangiectasias on the palms and face, masked facies, perioral skin tightening, muffled heart sounds, pulsus paradoxus, elevated JVD, and mild symmetric lower extremity weakness. Labs were notable for high titer +ANA with speckled and nucleolar/pattern, + anti-centromere antibody, and severe macrocytic anemia. Echocardiogram confirmed cardiac tamponade with right ventricular and right atrial collapse. MRI of the brain demonstrated bilateral occipital white matter hyperintensities, compatible with diagnosis of PRES. Her hospital course was complicated by refractory hypertension, bilateral pleural effusions, anasarca, and isolated myoclonic phenomena. Her cardiac tamponade was treated with pericardiocentesis. Our patient was treated with high dose corticosteroids and mycophenolate, her hypertension was controlled, and she had complete resolution of her seizures and altered mental status. Based upon the reversible nature of our patient’s neurologic symptoms and MRI findings, she was diagnosed with PRES.

Discussion: The pathophysiology of PRES is thought to be the result of either failed cerebral auto-regulation in the setting of accelerated hypertension, cerebral vasospasm, or endothelial dysfunction due to circulating toxins. Our patient had multiple risk factors that align with these theories, including hypertensive urgency, systemic sclerosis, cutaneous vasospastic phenomena, and use of immunosuppressive drugs. Although rare, given its reversibility with treatment, PRES should be considered on the differential diagnosis for any patient presenting with altered mental status and seizures who has a history of rheumatologic disease or immunosuppression, and medical providers should be aware of this etiological association.
Introduction: Scurvy is a rare condition in the Western world. The infrequency that it occurs in the US makes scurvy often missed or misdiagnosed initially, as it is not regularly considered. This is likely due to lack of knowledge of this disorder since it is uncommon. This case describes a patient with developmental delay secondary to cerebral palsy who develops a severe anemia due to dietary deficiency of vitamin C.

Case Description: The patient is a 39-year-old frail man with cerebral palsy and a seizure disorder, on baclofen, folate, vitamin B12, vitamin D, phenytoin and levetiracetam, who presented for pallor noted by his mother. She reported “spots” on his bilateral lower extremities two weeks before admission and gingival bleeding with a recently noted gingival “mass” by the dentist. Exam revealed tachycardia, mild hypotension, and a faint maculopapular rash with palpable petechiae on his bilateral lower legs. His hemoglobin was 6.4g/dL. This anemia was normocytic and hypochromic. There was improvement after transfusion, but the hemoglobin again downtrended. It was discovered on further questioning that the patient only ate canned vegetables and did not consume any fresh fruits or vegetables. He received a more varied diet while inpatient. The papular nature of the rash was gone by discharge, and the rash had faded almost completely. Additionally, there was a significant turn-around in the trend of the hemoglobin up to 9.5g/dL on discharge. He was started on 500mg bid of ascorbic acid. His vitamin C level came back after discharge at less than 8µmol/L, which is deficient at <11. On follow up, his repeat hemoglobin was 14.3g/dL at six weeks. At seven months his vitamin C level was up to 116µmol/L.

Discussion: This case illustrates the need for consideration of scurvy, a treatable and preventable condition, given the almost constantly pathognomonic presentation, to avoid unnecessary tests/consults. Scurvy is characterized by follicular hyperkeratosis and perifollicular hemorrhagic rash with corkscrew hairs, which occur early in the disease, which can be followed by anemia. While the mechanism of the anemia is undetermined, it is believed to result from blood vessel fragility, leading to gastrointestinal or soft tissue losses. Although, phenytoin is commonly known to be able to cause gum bleeding and gingival hyperplasia, it would not explain the anemia or the rest of the patient’s presentation. Scurvy results from lack of adequate dietary/supplemental intake of ascorbic acid, since humans lack the enzyme to synthesize vitamin C. Clinical manifestations do not occur until a person is deficient to less than 350mg of total body stores, which takes 2-3 months of zero ascorbic acid intake to achieve. Recognition of the condition is critical so appropriate treatment can be started to avoid possible catastrophic adverse events, including death.
Demographics, Clinical Characteristics and Quality of life in Homeless Population at Albuquerque Opportunity Center - 2014

Introduction: Over 3.5 million Americans experience homelessness every year and each year about 17,000 New Mexicans are homeless for at least part of the year. This includes single adults as well as families with children and unaccompanied youth. Without homes, these people also lose access to education, regular health care, employment and most of the things that many of us take for granted as part of our everyday lives. The most proximate cause of homelessness in America is poverty; Albuquerque’s poverty level is ranked the fifth highest in the nation. We postulated that the burden of psycho-social stress and high exposure to alcohol consumption and smoking and lifestyle in the homeless population will lead to increased risk of chronic conditions including metabolic syndrome and reduced QOL score. We also evaluated the corollary hypothesis to compare the clinical profile in homeless population with that of regular UNMHSC PCP run clinic population.

Methods: We worked at the shelter, at 715 Candelaria Rd NE, which is home to the UNMHSC student-led healthcare resource center. Fifty willing and able to consent homeless patient consented and completed evaluations including physical exam between Jan to June 2014. A comprehensive metabolic panel, lipid panel, urine microalbumin and HgbA1c were processed for each subject. We also administered a 35 item survey questionnaire and 16 item Q-LES-Q-SF to measure quality of life enjoyment and satisfaction. SAS statistical analysis was performed to identify the mean and standard deviation for the above variables. We are in the process to analyze the correlation analysis of our data to calculate association of clinical measures with the QOL.

Results: The mean age of the homeless cohort was 51 ± 8 yrs, all of them male with increased BMI and more than 50% were veterans. The clinical values were significantly increased for Glucose, A1C, AKP, AST, ALT, TChol, TG, and UACR compared to the PCP run clinic cohort. More than 50% of the homeless cohort patients consumed alcohol and smoked compared to regular PCP clinic cohort. The Q-LES-Q-SF score in homeless patient was very low in all items and overall percentage of QOL was at 40%.

Conclusion: We concluded that clinical profile in homeless cohort depicted a picture of metabolic syndrome and abnormal liver function. However comparing it to the clinical profile in regular PCP clinic suggested a normal metabolic and liver functions in PCP run clinic. This could be attributed to the successful counseling and aggressive management of glycemic control performed by the clinicians at the clinic. Another and perhaps more clinically significant conclusion is the unfortunate and incipient low score in QOL in the homeless group, despite the efforts at the center and the clinical support offered by UNMHSC.
Biopsy Confirmed Renal Injury in a Case of Levamisole-Induced ANCA-Vasculitis

Background: Levamisole is one of the most common adulterants of cocaine and has been isolated in approximately 80% of the U.S cocaine supply. Initially used as a chemotherapeutic agent, levamisole has significant immunomodulatory effects and a severe side effect profile. It has been associated with a classic clinical syndrome characterized by cutaneous purpuric lesions, arthralgias, leukopenia, and ANCA-positivity. However, the spectrum of disease is wide and can include cardiovascular, pulmonary, neurologic, and renal manifestations. Limited data is available regarding renal injury caused by levamisole toxicity, and only one kidney biopsy has been reported in the literature. We present a case of biopsy-proven pauci-immune glomerulonephritis in an active cocaine abuser with the vasculitic syndrome of levamisole toxicity.

Case Report: This patient is a 65-year-old man with a history of hepatitis C virus, type 2 diabetes mellitus, and active cocaine abuse who presented with non-healing skin lesions. His last cocaine ingestion was reportedly 2 weeks prior. His creatinine was elevated at 3.42 mg/dL (baseline 2 years prior was 0.9). ESR was 111 mm/hr and CRP was 1.6 mg/dL. ANA titer was 1:320, p-ANCA titer was 1:10240, anti-MPO was elevated. Skin lesions were most notable in bilateral upper and lower extremities, trunk, upper back and bilateral ear lobules. Biopsies of 2 sites revealed thrombotic vasculopathy consistent with levamisole-induced vasculitis. Given concern for vasculitides and contributions from other processes (i.e., diabetes mellitus), the patient underwent renal biopsy. Renal biopsy revealed focal endocapillary, pauci-immune glomerulonephritis. Features of cryoglobulinemic glomerulonephritis were not identified. Creatinine remained elevated after discharge with new baseline between 3-3.7 mg/dL and GFR <30, suggesting CKD.

Discussion: Levamisole has been reported to potentiate the stimulant effects of cocaine, acting both synergistically and additively on acetylcholine and dopaminergic receptors. Its immunomodulatory effects result in an ANCA-positive syndrome characterized by autoantibody formation leading to diffuse vasculopathy and a variety of systemic manifestations. Of these manifestations, levamisole-induced renal dysfunction is an emerging entity, which remains poorly described. A case report published in April 2014 recognized a patient with likely, although not biopsy-proven, AKI secondary to levamisole toxicity. Prior to this there was one report of nephrotic syndrome induced by levamisole-adulterated cocaine, but confirmatory renal biopsy was lacking. The only documented biopsy-proven case of pauci-immune focal glomerulonephritis associated with levamisole-adulterated cocaine was published in 2011, and there has been limited data since. Our case is unique in that both skin and renal biopsies were performed, both of which demonstrated histological consistency with an underlying levamisole-mediated process. Thus, this report highlights further the need for practitioners to be cognizant of levamisole-adulterated cocaine as a potential cause of both AKI and CKD in select patient groups presenting with other classical findings of levamisole toxicity.
A "Shock"ing Twist

The diagnosis of shock encompasses a variety of distinct entities including septic, hypovolemic, cardiogenic, anaphylactic, and neurogenic shock. Despite all having the similar end result of inadequate oxygen delivery to tissues and vital organs progressing to end-organ dysfunction, the pathophysiology of each type of shock is different and early identification is essential to effective management. A 43 year-old female presented to the emergency department with a 4-day history of diffuse, progressive abdominal pain associated with vomiting and diarrhea. She was afebrile, tachycardic (pulse 125) and hypotensive (94/65). Labs revealed WBC 23, hemoglobin 11, HCO3 7, Creatinine 6.02, lactate 4.1, and pyuria on urinalysis. CT of the abdomen demonstrated fluid in the cul-de-sac, multiple loops of inflamed bowel, and phlegmonous tissue in the upper pelvis. A central line was placed and IV fluids, levophed, and empiric antibiotic therapy were started. Despite aggressive resuscitation, the patient worsened, becoming progressively more obtunded and developed a reticular rash. On further questioning, the patient noted that her pain began simultaneously with her menses, and reported insertion of a tampon three days prior without removal. Pelvic exam revealed purulent vaginal discharge with no visible tampon. Blood cultures resulted positive for Streptococcus pyogenes. The patient was taken to the operating room for an exploratory laparotomy with findings of edematous ovaries and tubes, and purulent peritonitis requiring total hysterectomy with bilateral salpingectomy. The patient was diagnosed with toxic shock syndrome and unfortunately expired thereafter with refractory shock and multiorgan failure.

Toxic shock syndrome (TSS) is an acute systemic illness associated with superantigen-producing strains of Staphylococcus aureus and Streptococcus pyogenes. In the literature, menstrual TSS has generally been associated with S. aureus. Although tampon-related TSS is typically associated with S. aureus infection it can also be caused by isolated S. pyogenes which in this case was not an expected result. The classic presentation of TSS in an abrupt onset fever, vomiting, diarrhea and abdominal pain, macular rash, sore throat, and myalgia followed by hypotension and multiple organ failure. This case emphasizes that common diseases present commonly, although TSS has been less recognized in recent years owing to its infrequent occurrence. This is likely what led to the delay in diagnosis. Early diagnosis is critical as hypotension and multiple organ failure can occur rapidly with potential for resistance to aggressive fluid and antibiotic therapy.
An Interesting Case of Severe, Multifactorial Hypokalemia

Hypokalemia is common in hospitalized patients, and it is easily treated with identification of the underlying etiology. In some patients, however, the etiology is not obvious or it is multifactorial, therefore creating a challenge. A 56 year-old female presented to the emergency department ten days after sustaining a splash burn to her left foot. She was admitted to the burn service for wound care. Shortly thereafter, she was transferred to the medicine service due to findings of a serum potassium of 1.8mg/dL along with an oxygen saturation of 83% on room air, alkalemia with HCO3 of >45mg/dL, and tachycardia up to 128bpm. Her only known medical problem was a seizure disorder secondary to head trauma sustained 20 years ago for which she was taking two unknown medications. She was a 20+ pack-year smoker, but denied alcohol or drug use. After being admitted, she was observed to have nausea, vomiting, and tremors; she also admitted to unintentional weight loss, fatigue, heat intolerance, increased appetite, and diarrhea. With further work-up, her problem list grew to include: hypokalemia, metabolic alkalosis, respiratory acidosis, volume depletion, peptic ulcer disease, severe pyloric stenosis, COPD, malnutrition, tachycardia, hyperthyroidism, heart failure, anemia, pulmonary hypertension, mitral valve regurgitation, and delirium, among others. The consequences of her hypokalemia were minimal, with slight muscle weakness and a mildly prolonged QT interval. Her serum potassium corrected from 1.8mg/dL to 4mg/dL over 48 hours, after replacement with potassium chloride. Further management included treatment of her: volume depletion-with intravenous and oral fluids; peptic ulcer disease- with proton pump inhibitor; hyperthyroidism- with anti-thyroid medication and beta-blocker; COPD- with inhaled steroid, long-acting beta-agonist, anti-cholinergic, and supplemental oxygen. Unfortunately, after five days in the hospital, she refused further workup secondary to her desire to return home, and she was discharged in stable condition.

The etiologies of hypokalemia can be divided into: transcellular shifts, GI losses, renal losses, and inadequate intake. In this case, the patient had: metabolic alkalosis causing an intracellular shift through cellular K/H+ exchange pump, volume depletion causing renal losses, hyperthyroidism causing a shift through Na/K ATPase, upper and lower gastrointestinal loss, and inadequate dietary intake. If a clear source is not identified by history alone, then more investigations should be undertaken: acid-base status, magnesium levels, urinary potassium studies, and serum renin/aldosterone concentrations. Treatment is guided by etiology and severity; the goal is to prevent complications and address the underlying cause. Potassium repletion can be achieved with intravenous and oral preparations, and requires close observation until stabilization. Though it was not known on initial presentation, this patient clearly had a multifactorial explanation for her low potassium level. She responded well to oral and IV potassium replacement, in combination with treatment of her various underlying conditions.
Effectiveness of Thrombophilia Testing: Testing To a Fault?

Introduction: Thrombophilias can occur from a variety of inherited and acquired abnormalities. Patients with these abnormalities of the coagulation pathway often have a higher propensity to develop venous thromboemboli (VTE), although the risk for developing a VTE is not dependent on the existence of an acquired or genetic abnormality. The question then remains, in the event of a VTE, when is testing indicated for a potential thrombophilic pre-disposition? The standard reasons to consider testing are to: a) Look for an underlying cause of an unprovoked VTE; b) Assess the probability of a repeat event thereby guiding duration of anticoagulation therapy; and c) Identify asymptomatic family members with an underlying predisposition to thrombophilia who might benefit from thromboprophylaxis or genetic counseling. In an effort to improve testing efficiency, the hypercoagulable panel order set (HCPAN), a set of 8 tests indicated for inherited thrombophilia, was instituted at the University of New Mexico at the outset of induction of the electronic medical record. However, we hypothesize that the HCPAN is being utilized outside of guideline-directed diagnostic utility.

Methods: A list of 600 HCPAN tests from October 2012 to November 2013 was generated from the TriCore historical database. This included all adult inpatient and outpatient instances. The investigators conducted manual chart review of each patient, evaluating risk factors for VTE, the rationale for ordering the HCPAN, and the level and service of the ordering provider. The protocol was approved by the Institutional Review Board for the UNM School of Medicine.

Results: Of 50 patients analyzed, 50% were categorized as having a provoked versus unprovoked VTE, while the other 50% had no listed reason for ordering the HCPAN or had a reason unrelated to the VTE. Of those listed as provoked vs unprovoked, 54% of those were ordered for a provoked VTE. Various departments order the HCPAN at differing time intervals, including when acute VTE are present, and in the setting of active anticoagulation. The data also indicate that at least 40% of the tests were ordered in the presence of an acute VTE.

Conclusions: In summary, the data indicates that the majority of HCPAN tests are being ordered outside of recommended guidelines, either because they are being ordered for a provoked VTE, when less than the 8 included tests are indicated, or in the presence of an acute VTE. In addition, the rationale for ordering the entire HCPAN was often not documented. This establishes that hypercoagulability testing at UNM Hospital is an area which may benefit from a quality improvement intervention.
Hashimoto’s Encephalitis - A Rare Entity

Hashimoto’s encephalitis is a rarely encountered, controversial syndrome with an unknown pathophysiology. It is characterized by sub-acute confusion, seizures and myoclonus thought to be immune-mediated rather than a direct effect of an abnormal thyroid state on the nervous system, with high serum antithyroid antibodies. Since 1966, there have been only 121 cases of this disease. Here we present a case of Hashimoto’s encephalitis.

Case Report: A 70-year-old female with a history of hypothyroidism presented with altered mental status attributed to hypertensive encephalopathy and was intubated for airway protection. Her TSH level was greater than 100 and free T4 was 1.1. After successful extubation, she remained somnolent and communicated very minimally with the medical staff. A GCS score was determined to be 7. A complete work-up including medicine reconciliation, basic laboratory analysis, neuroimaging, lumbar puncture, EEG, and cultures were unremarkable, although EEG showed abnormal cerebral activity. As her mental status did not improve, anti-thyroglobulin antibodies were checked and found to be 68 and a thyroid peroxidase antibody level was greater than 1000. The patient was started on intravenous T3 and Prednisone and her mental status slowly improved with complete normalization of her GCS score and communication abilities.

Discussion: Hashimoto’s encephalitis is a rare syndrome associated with Hashimoto’s thyroiditis. First described in 1966, there have only been 121 cases reported in the medical literature and the underlying mechanism remains unknown. Clinically it is characterized by altered mental status, seizures, and myoclonus. Studies show that Hashimoto’s encephalitis is unrelated to the state of functioning of the thyroid gland, and is secondary to an autoimmune/inflammatory process. Elevated anti-thyroglobulin antibodies and/or anti-thyroid peroxidase antibodies, a compatible clinical presentation and a response to corticosteroids defines the syndrome. Our case illustrates a patient who presented with altered mental status, an elevated TSH, a moderately decreased free T4 level and abnormal cerebral activity on EEG. The differential included myxedema coma, although she did not present with hypothermia, hyponatremia, or hypercapnia, hallmarks of the disease and had anti-thyroid and anti-thyroid peroxidase antibodies in her serum. Myxedema coma, a rare and fatal disease, has a poor prognosis without aggressive treatment. The patient improved with intravenous T3 and Prednisone, an atypical treatment regimen for myxedema coma. The significance of our case is that it describes a patient with a constellation of symptoms, findings and clinical course suggestive of Hashimoto’s encephalitis, which are usually attributed to other thyroid disorders. The rarity of the syndrome makes it difficult to identify. However, it is important for physicians to understand it and be aware of its existence to avoid misdiagnosis and ascertain appropriate management for this as well as for other more aggressive and fatal thyroid-related causes of encephalopathy.
Os Trigonum Syndrome In a Patient With Ankle Pain

Introduction: The differential diagnosis of posterior ankle pain includes a variety of inflammatory and non-inflammatory conditions that can be diagnosed with a careful physical and radiographic examination.

Case description: A 44 year old African-American woman with a history of primary hyperparathyroidism, beta-thalassemia trait, and previous spinal fusions at multiple levels for disc protrusions presented with a 3 year history of unilateral left ankle swelling. The swelling was exacerbated by physical activity and she had no history of any injury to the ankle. Clinical examination revealed mild swelling with tenderness of the postero-lateral aspect of the ankle, bilateral lower extremity muscle weakness at the hip flexors, and weakness in the anterior and lateral compartments of the left lower extremity. Laboratory investigations were notable for low-level ANA but no specific autoantibody subfamilies, and negative inflammatory markers. Rheumatological workup did not support generalized inflammatory arthropathy, crystal induced disease, advanced osteoarthritis, or parathyroid arthropathy. Ligament injury was suspected and an ankle X-ray and MRI was obtained. MRI scan revealed a well corticated, triangular bone posterior to the talus, consistent with an os trigonum accessory bone with associated tenosynovitis of the flexor hallucis longus tendon (FHL). The imaging also revealed Achilles tendon enthesopathy at the calcaneus, generalized soft tissue swelling of the ankle, prior partial tear/sprain of the anterior talofibular ligament (ATFL), and a remote medial malleolar avulsion injury (Fig. 1 X Ray, Fig. 2 MRI). The patient was treated with brace immobilization and physical therapy with good response.

Discussion: This case illustrates the need of a broad differential diagnosis in patients with ankle pain especially in patients with unilateral complaints and no history of trauma. Os trigonum is a small bone of the postero-lateral aspect of the talus formed from separate ossification centers, which fails to unite with the talus itself. This is a rare and uncommon cause of non-inflammatory ankle pain, with an estimated incidence of 3-10%, and when symptomatic, it is characterized by recurrent postero-lateral ankle pain with swelling and stiffness of the posterior ankle. It is commonly symptomatic in ballet dancers, or people who participate in sporting activities that require forced plantar flexion. Frequently missed diagnosis can lead to unnecessary conservative or surgical interventions with no improvement in symptomatology and patient frustration. This is a rare but important non-inflammatory cause for ankle pathology that should be considered in the differential diagnosis of unilateral ankle pain.
PTSD Mouse Model: Validation of Cognitive Defects in Predator Exposure Paradigm

Posttraumatic Stress Disorder (PTSD) is caused by exposure to a traumatic event in which an individual experiences a situation that is life-threatening or wherein the individual feels powerless over his or her situation. PTSD is a prevalent, debilitating illness affecting approximately 8% of the U.S. population, with approximately twice as many women affected. Many behavioral characteristics exhibited by people with PTSD can be observed in mice following a predator exposure paradigm. This paradigm appears to be perceived by the mice as life-threatening, rather than just stress-inducing or painful, and thus a PTSD-like state is induced. It has been shown that PTSD alters cognitive function in individuals. It is the goal of this study to employ a mouse model of PTSD to recreate, observe, and perhaps further understand the level of cognitive impairment involved in PTSD. This will be done by studying cognition in PTSD-like mice using a trace-conditioned learning and memory test, as well as a fear extinction procedure. The results of this study provide insight into the utility of the PTSD-like mice in studying PTSD-related cognitive impairment and further the understanding of PTSD-related cognitive deficits. Our results show the predator odor-exposed mice displayed increased fear leaning/memory, as assessed by measuring their freezing response to the conditioned stimulus (the tone). This may model hypervigilance, a state of heightened awareness of environmental stimuli, that is seen in PTSD patients.
Sweet’s Syndrome and Insecticides: A Cautionary Tale

Sweet’s syndrome is a rare condition characterized by fever, neutrophilia, tender erythematous skin lesions, and a diffuse neutrophilic infiltrate of the upper dermis. Its classic presentation is seen in women ages 30-50 with fever, rash, and without neurological symptoms. Etiologies include medications, malignancies and connective tissue disorders. We present a case of Sweet’s syndrome with neurological features due to an unusual etiology (insecticide). A 50 year old Hispanic male with past medical history of polysubstance abuse presented with a vesiculomaculopapular rash at varying stages of healing along his neck, trunk and upper extremities, and a nontender erythematous papular rash along the extensor surface of the legs for two weeks. Prior to admission, he was seen at an urgent care clinic and was sent home with Permethrin cream, Allegra, and Bactrim. The following day he applied the cream to his lesions but developed new lesion eruptions and periorbital edema. He presented to the ED and rapidly developed altered mental status. His physical exam was significant for systemic inflammatory response, nonfocal neurological exam, negative guaiac stool and DRE, and umbilication and eschars in many of the papular lesions. Laboratory studies revealed pancytopenia but no evidence of infection. Patient was admitted to the hospital and dermatology and infectious disease consults were obtained. CT and MRI of brain revealed no acute intracranial abnormality; EEG showed a nonspecific moderate diffuse encephalopathic process. He tested negative for numerous bacterial, viral, and fungal infections by tissue, wound, blood, urine, and CSF analyses. Obtained punch biopsies of the lesions returned positive for Sweet’s Syndrome. On the 4th day, the patient was able to communicate and stated he had sprayed Raid, an insecticide, on his body for several days to deter mosquitos. Toxicology concluded the manifestation of Sweet’s Syndrome to be consistent with exposure to permethrin-containing insect repellent, exacerbated by the permethrin cream utilized the day before admission. His mental status improved and he was discharged on the 9th day with topical prednisone and 60 milligrams of oral prednisone to be taken daily, tapered by 10 mg weekly over 6 weeks. This case is unique in not only in the unusual patient characteristics and etiology of Sweet’s syndrome, but as well as the transient encephalopathic component that has been seen only in a few cases.

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is a rare but potentially life threatening Type IVb Hypersensitivity reaction which occurs 2-8 weeks after administration of the offending agent and manifests as multiorgan disease, diffuse maculopapular rash and lymphadenopathy, and hematologic derangements including eosinophilia. The infrequency with which it is encountered, the time-lag from drug administration to manifestation, and the variety of clinical presentations/organ system involvement make it a challenging diagnosis, especially with a rarely implicated agent such as Bactrim.

A 55 year old morbidly obese pre-diabetic male presented to the UNM Urgent Care with a scalp abscess found to be MRSA positive, and was prescribed a ten day course of Bactrim (Sulfamethoxazole/Trimethoprim). One week later, he presented to UNM-ED with fevers, chills, and general malaise and was discharged without admission after negative CT scan of brain ruled out infectious spread. Three days later the patient returned to the ED with a diffuse maculopapular rash, eosinophilia, leukopenia with thrombocytopenia, and elevated transaminases and was admitted for likely sepsis secondary to prior scalp abscess and drug reaction to Bactrim which was switched to doxycycline. He was discharged after two days. Two days later he returned to the ED with dyspnea and hypoxia and was diagnosed at that time with Healthcare Associated Pneumonia given his recent hospitalization and chest X-ray findings, while his diffuse rash persisted. The patient’s respiratory status continued to worsen, and he was transferred the next day to the MICU and was intubated. Given the concern for HCAP vs MRSA sepsis, an extensive infectious work-up was completed, which was negative. The patient was then diagnosed with DRESS five days after intubation, and three weeks after first being treated with Bactrim. The patient remained intubated for 16 days before being extubated, and is currently stable and clinically improving.

This case highlights the challenging nature of DRESS syndrome; non-specific signs and the time-lag before onset make DRESS a challenging clinical mimicker to diagnose. Because the various DRESS criteria presented at different times in this patient, and Bactrim is not recognized as a major cause of DRESS, diagnosis was further impeded. Due to these challenges, as well as the potentially life-threatening severity of the disease, it is important for providers to have a high index of suspicion in a patient with potential DRESS, and to consider this diagnosis on the differential. This case further underlies the importance of performing a med-reconciliation at every hospital visit, given the delay in presentation of DRESS. Finally this case serves as a great learning tool for familiarizing oneself with the criteria needed to make a diagnosis of DRESS.
A Case of Acute Eoinophilic Pneumnia

Introduction: Idiopathic Acute Eoinophilic Pneumnia (IAEP) is a rare subset of acute respiratory failure marked by hypoxemia, diffuse pulmonary infiltrates and pulmonary eosinophilia. This was first described in 1989, since retrospective and case reports have supported the hypothesis that AEP is an acute hypersensitivity reaction. Generally, AEP presents after exposure to an inhaled agent a healthy individual. Exposures have included smoking, moving soil, and some medications, specifically antibiotics. This usually affects males more often than women, and young individuals. Here we present a case of AEP after an exposure to pet urine in a patient with no previous cat allergy. This is the first case reported in association with animal allergens or urine exposure.

Case Presentation: A previously healthy 31-year-old man, who was an electrician, presented to the emergency department with a 7-day history of non-productive cough, exertion dyspnea, subjective fevers and nightsweats. He had been removing carpet, which was saturated with feline urine. On admission, he presented with acute reparatory failure, which required 6L of O2 by face mask to maintain saturations >90%. His WBC counts were 19,400/μL with 68% neutrophils and 12% eosinophils. Serum IgE was elevated at 2,996 int units/dL (Normal 0-87). An extensive examination of serum antibodies or cultures of sputa proved negative for bacterial, viral, and fungal infection. Chest CT showed diffuse infiltrates. A bronchoscopy examination was performed on day 2. BAL contained an eosinophil count of 82%. Based on these findings, the patient was diagnosed with AEP. The eosinophil fraction of the peripheral WBC count had peaked and started to decrease on hospital day 3 after steroid treatment with 60mg of methylprednisolone was initiated. At follow up visits he continued to be asymptomatic.

Discussion: Many cases of AEP have been reported in association with occupational or environmental exposures. There hasn't been a report in association with the inhalation of animal allergens or urine exposure and AEP. IgE mediated reactions are common in laboratory staff workers exposure to rat urine, and were more common in those workers with other atopic allergies or high serum IgE counts prior to exposure. Feline urine has been studied which found that feline urine had two allergens present, which were different from feline saliva or pelt.